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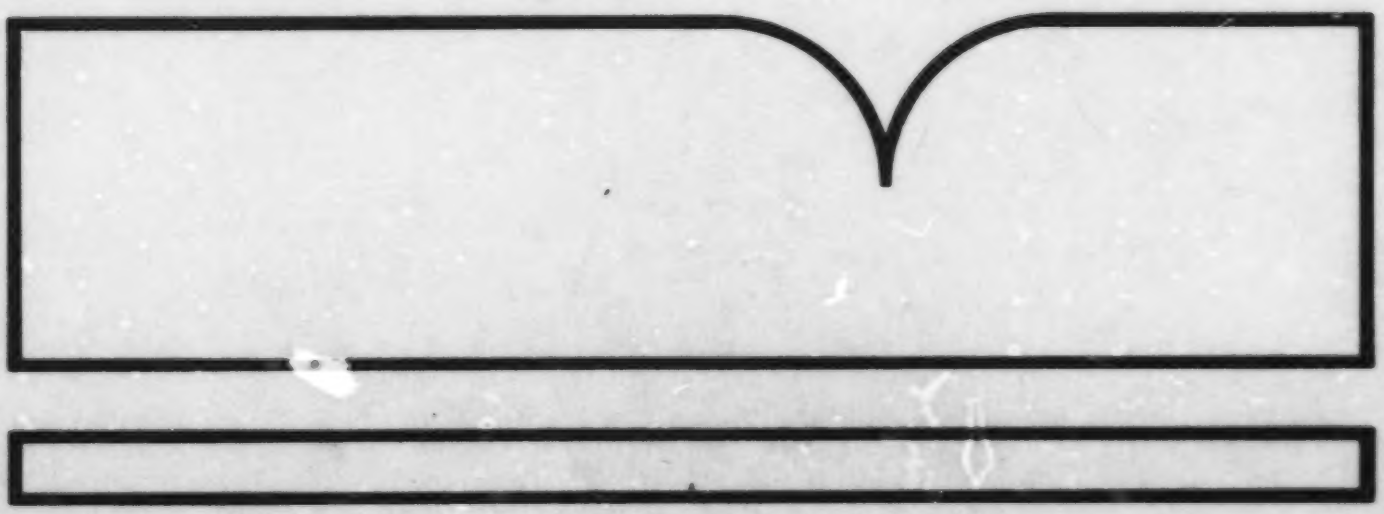

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Health effects associated with wastewater treatment and disposal

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WASTEWATER TREATMENT PLANTS

In an epidemiological study of viral hepatitis in Worcester, Mass., Hoffman *et al.*[1] found significantly higher incidences of the disease in those areas of the city served by old water and sewer pipes, combined sewers, and low pressure water distribution systems. Rylander *et al.*[2] reported on fever caused by exposure to *Flavobacteria* endotoxins from a humidifier, similar to wastewater treatment plant worker exposures on which they had previously reported. The health effects on treatment plant workers of the dumping of a large quantity of the industrial chemical hexachlorocyclopentadiene into a municipal wastewater system were described by Morse *et al.*[3] They found eye irritation, headache, throat irritation, proteinuria, and elevated serum lactic dehydrogenase; findings were absent after 3 weeks. It was concluded that wastewater treatment plant workers are particularly vulnerable to exposure to toxic industrial chemicals discharged into wastewater systems.

Carnow *et al.*[4] performed an extensive study of the health effects of aerosols emitted from an activated sludge plant, and concluded that: the wastewater treatment plant was a source of airborne total aerobic bacteria-containing particles and total coliform bacteria as measured in the community; the concentration profiles of trace metals, particulates, nitrates, sulfates, sulfur dioxide, nitrogen dioxide, hydrogen sulfide, and chlorine in the community indicated that the plant was not a source of these materials; ambient concentrations of bacteria showed no obvious correlation with plant operating characteristics or wastewater concentrations; bacteria concentrations did not display obvious trends with solar radiation, temperature, and relative humidity; virus and coliphage measurements in wastewater and air were inadequate in number to determine their concentrations with any confidence; and significant linear relationships were not found by regression analyses of total viable particle exposure indices and the following health measurements: self-reported acute illness rates, pathogenic bacteria isolation rates, prevalence rates of antibody to certain enteroviruses, and virus antibody titers.

Johnson *et al.*[5] conducted an aerosol monitoring program at an advanced wastewater treatment facility using the activated sludge process, recently constructed next to an elementary school. Although a relatively insensitive measure of illness, attendance at the nearby school, when compared to eight control schools, provided no evidence of adverse effects from the wastewater treatment plant operation. If any adverse effect had occurred, it was slight enough to be completely obscured by the usual school absenteeism factors. The students had probably received a peak daily dose of about 9 cfu of mycobacteria and 3.5 cfu of fecal streptococci, which might only be 100 to 1 000 times the usual outdoor background dose. A recent symposium on the health risks of wastewater aerosols was summarized.[6] The general conclusion was that these aerosols did not appear to present a measurable health risk to plant workers or nearby residents.

COMPOSTING

A comprehensive report[7] from a workshop on the health and legal implications of wastewater sludge composting concluded that pathogenic risks posed by composting and compost distribution were low enough to permit development and operation of municipal composting systems, provided simple precautions were adopted. The major results of the workshop were summarized by Marcus *et al.*[8] Marsh *et al.*[9] performed an extensive review of the recent literature on the health effects of *Aspergillus fumigatus*, a common fungus developing during composting, covering the nature of aspergillosis, clinical observations, epidemiology, and risk assessment and control. Fraser *et al.*[10] calculated the U. S. incidence rates in

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1976 of systemic mycoses necessitating hospitalization, finding: histoplasmosis (23.0 cases per mil per yr), coccidioidomycosis (17.9), aspergillosis (4.8), actinomycosis (3.2), cryptococcosis (2.3), candidiasis (1.8), sporotrichosis (0.5), and blastomycosis (0.2). Patients with aspergillosis had high prevalences of leukemia or lymphoma, other malignancies, and chronic bronchitis or emphysema. The incidence of aspergillosis had increased by 158% since 1970; increasing numbers of persons with immunosuppressive conditions and aging of the population were thought to have contributed to the increase. Malo and Paquin [11] found a 21.5% prevalence of immediate sensitivity to *Aspergillus fumigatus* in Canadian asthmatics, and concluded that the prevalence in North America was at least equal to that found in Great Britain. Staib *et al.*[12] reported on finding *Aspergillus fumigatus* in the soil of ornamental plants, and discussed the epidemiological significance of this to susceptible persons.

LAND APPLICATION OF WASTEWATER AND SLUDGE

Jewell and Seabrook [13] reviewed the history of land treatment of wastewater, discussing why land application of wastes evolved into the main treatment alternative in the 1800s, why it was subsequently relegated to a disposal alternative, and why it was now being rejuvenated as the most effective pollution control alternative. The role played by human disease in these historical changes is included. Fitzgerald [14] briefly reviewed his own and other research on the potential health effects of the application of anaerobically digested sludge to land, and subsequent livestock grazing. He concluded that, while the increase in trace element levels in soil may be great, the increase in plant levels was much less, and that in animal levels (aside from specific organs) much less yet. Transmission of pathogenic organisms in raw wastewater, through appropriate processes to anaerobic sludge, then to forage and finally to animals, was remote. Pahren *et al.*[15] appraised the health risks associated with land application of municipal sludge, particularly with respect to cadmium, and concluded that such sludge should contain less than 60 ppm cadmium and the soil should be maintained near neutrality in those situations where food is grown. In geographical areas where parasites normally were found, the addition of municipal sludge to agricultural land would be expected to enhance the risk of transmission to man. Sagik *et al.*[16] briefly reviewed the public health aspects of the land application of municipal wastewater effluents and sludges, suggesting that current treatment chain design, appropriately used, was probably adequate to remove much of the potential public health hazard caused by wastewater effluents. They emphasized the importance of finding the minimal infectious dose of waterborne pathogens and levels which would induce overt disease in a healthy human being. An overview of land application of wastewater and sludge was given by Wright and Rovey,[17] emphasizing siting for the protection of public health. Included was an account of the Bortnichy State Farm at Kiev, USSR, where potatoes and other crops for human consumption have been raised for more than 14 years using secondary effluent, without any harmful health effects.

The research program at New Mexico State University and Sandia Laboratories on using raw wastewater solids as feed for cattle was reviewed by Smith *et al.*[18] Iron, lead, and pesticides were elevated in experimental animals, but risks to animal health and to the quality of meat products were considered to be low. It was concluded that the potential economic, social, and environmental benefits from the use of wastewater solids as supplemental feedstuffs for cattle could be substantial, and that more rigorous investigations to assess fully the risks of the practice are warranted.

A portion of the Conference on Risk Assessment and Health Effects of Land Application of Municipal Wastewater and Sludges, held in December 1977 at San Antonio, Tex., was summarized briefly.[19] The problems of performing risk analyses, with special reference to sludge management, were discussed by Jutro,[20] who emphasized the incompleteness of the informational foundation upon which decisions must be based. Jutro [21] also briefly described an ongoing project to develop a risk assessment methodology for sludge management decisions.

The federal groundwater protection program was briefly reviewed by Kimm,[22] EPA's deputy assistant administrator for drinking water. The final federal regulations, required by the Resource Conservation and Recovery Act of 1976 and the Clean Water Act of 1977 governing the land application of municipal wastewater sludge and other nonhazardous solid wastes,

appeared.[23] These regulations were briefly summarized by Easton.[24]

Parasites, bacteria, viruses. The Chicago MSD and IIT Research Institute [25, 26] reported on viral and bacterial levels resulting from the land application in Fulton County, Ill., of municipal sludge which had been anaerobically digested for 14 days, barged for 200 miles, and lagooned for at least 60 days. They found no detectable viruses in the sludge, and no effect of the land application of sludge on the bacterial and viral quality of the surface water, runoff, or groundwater. Sheridan *et al.*[27] described an ongoing project to develop a computer model to predict pathogen survival during sludge treatment and subsequent environmental movement of the pathogens to man, using several sludge utilization alternatives. Sorber *et al.*[28] discussed the potential public health effects of municipal wastewater spray irrigation aerosols, concluding that significant numbers of microorganisms were aerosolized, but that the associated risks to man were unclear. Reed [29] took issue with statements that wastewater aerosols from land application presented a health hazard that justified a very low coliform limit. He suggested low pressure, downward-spraying nozzles and restricted public access. He also questioned the need for the 2.2 total coliform limit for food crop irrigation and recreational impoundments. Crook [30] replied that the California treatment requirements for surface irrigation of food crops were much less stringent than those for spray irrigation. The requirement for reclaimed water used to surface irrigate food crops did not provide a virus-free effluent. Craun [31] reviewed the outbreaks of waterborne disease in the U. S. during the period 1971-77, noting that a significant number of outbreaks and illnesses occurred because of the lack of treatment of ground water and because of the interruption of disinfection and inadequate disinfection of contaminated ground water.

Fitzgerald [32] reviewed the potential impact on public health of parasites in soil/sludge systems, concluding that the proper utilization of wastewater sludge did not pose any great threat to the health of society through actual transmissions of pathogens that might be present in sludge. Lindquist [33] found a high rate of survival of *Ascaris suum* eggs in pigpen sludge in aerobic pits, but no *Ascaris* eggs in pasture soil to which the sludge had been applied for several years. He concluded that landspreading was a safe way to dispose of pit sludge and effluent. The increasing incidence of toxocariasis (visceral larva migrans from dog and cat roundworm) was briefly reviewed,[34, 35] and traced to the dog population of 80 million in the U. S., with a *Toxocara canis* infection rate of 20 to 50%. The parasite's eggs are transferred with dog feces to soil, sandboxes, and other points of human contact, where they remain infective for months or years. Upon ingestion by humans, they hatch, and the larvae penetrate the intestinal wall and migrate in the bloodstream to other organs. Preliminary serological screening has shown infection rates of 0 to 13% in several American populations. Fox and Fitzgerald [36] examined Chicago wastewater and sludge for parasites, finding up to 500 Giardia cysts per litre in raw wastewater, and no Giardia in anaerobically digested sludge. Giardia cysts were most prevalent in primarily domestic wastewater, especially during the warm months. In reexamining early studies of the infectivity of *Giardia lamblia* to man, Rendtorff [37] showed that a single cyst failed to infect healthy men, but that doses of 10 to 1 mil cysts produced 100% infection. Cysts survived for at least 16 days in tapwater at 8°C, and it was concluded that water was a very efficient vehicle for the transmission of Giardia, and small numbers of cysts could readily produce infections in a susceptible host. The waterborne outbreaks of giardiasis in the U. S. for 1972–77 were reviewed by Craun,[38, 39] who concluded that disinfection alone was ineffective as treatment of surface water sources, and that negative coliform tests did not provide assurance that water was free of Giardia cysts. Teutsch *et al.*[40] reported on an outbreak of 37 cases of toxoplasmosis in patrons of a riding stable in Georgia; infection probably occurred by ingestion of *Toxoplasma gondii* cysts from the feces of infected cats, either through inhaled dust or contaminated food. The 95% rate of clinical disease among infected persons and the low rate of original clinical diagnoses indicated a substantial underdiagnosis of the disease and suggested that toxoplasmosis was a more common cause of clinical illness than previously recognized; the authors noted that 30% of the population of Georgia had toxoplasma antibodies. Lobel and Kagan [41] discussed the methodology and interpretation of seroepidemiological investigations of parasitic diseases, with applications to those caused by *Entamoeba histolytica*,

Toxoplasma gondii, and *Echinococcus granulosus*.

Baubinas and Vlodavets [42] detected bacterial aerosols 250 to 600 m from a wastewater spraying apparatus in the Soviet Union, with intestinal pathogens recovered from the surface of grass and in aerosols at 200 to 400 m. An outbreak of enteritis caused by *Campylobacter fetus* subsp. *jejuni* was reported,[43] and it was felt that this bacterium might prove to be as common a cause of diarrhea as Salmonella and Shigella.

The fate of viruses in the land application of wastewaters and sludges was extensively review by Dubois *et al.*[44] It was concluded that the present level of knowledge provide an inadequate basis for assessing the risks posed by human enteric viruses in wastewater effluents and sludges applied to land. While certain data had indicated prolonged survival of enteroviruses in soils and had identified particular conditions that were conducive to enterovirus movement within the soil matrix, the extent to which these data could be generalized to predict the fate of viral pathogens in soils other than those tested was uncertain. Systematic study involving more diverse soil types was needed. In addition, the impact of long-term use of any soil system for disposal of effluents or sludges upon the capacity of that soil to remove viruses effectively must be ascertained. Even when virus movement into ground water was demonstrated, a consensus concerning the extent of risk might be precluded by the lack of definitive data indicating viral doses required for infection by the water route. Goyal and Gerba [45] studied the comparative adsorption of a number of different types and strains of human enteroviruses and bacteriophages to nine different soil types. They concluded that, in spite of the fact that most of the studies on virus adsorption to soils had utilized poliovirus as the model system, no one enterovirus or coliphage could be used as the sole model for determining the adsorptive behavior of viruses to soils and that no single soil could be used as the model for determining viral adsorptive capacity of all soil types. Scheuerman *et al.*[46] examined the transport of viruses through organic soils and sediments, and concluded that organic soils were not quite suitable for the application of wastewater effluents because of the demonstrated interfering effect of water-soluble "humic substances" with viral retention. The subject of viruses in the water environment was briefly reviewed by Mahdy.[47] Melnick and Gerba[48] briefly brought attention to the risk of spreading enteric viral disease by the water route as a result of increased wastewater recycling, low treatment inactivation of viruses, and traditional indicator bacteria not reflecting viral densities. Katzenelson and Kedmi [49] indicated that poliovirus was unsuitable as an indicator of the virological quality of potable water. The diagnostic tests, immunity aspects, and epidemiology of the agents of viral gastroenteritis were extensively reviewed by Holmes.[50] These agents are now considered to be primarily rotovirus and parvovirus-like agents, as well as a group of less important viruses. Brandt *et al.*[51] examined fecal samples from 804 Washington, D. C., children (up to 2 yr) with gastroenteritis, and found that 35% were shedding human rotavirus. The prevalence of rotavirus was highest in January. Wenman *et al.*[52] performed a prospective study of rotavirus infections in 98 families with newborn children, and found infections in 36 of 102 adults whose children had rotavirus infection as compared with 4 of 86 without infected children. They concluded that rotavirus was a mild but common infection in parents of young children. Greenberg *et al.*[53] studied serologically 25 separate outbreaks of nonbacterial gastroenteritis, found 8 of 25 to be related to the Norwalk virus and 1 to rotavirus, and concluded that parvovirus-like agents played an important role in epidemic nonbacterial gastroenteritis in adults and older children. Richmond *et al.*[54] reported an outbreak of gastroenteritis in young children, caused by adenovirus. They noted that advenovirus have been demonstrated by microscopic techniques in 5 to 12% of stool specimens from young children and babies with acute gastroenteritis, and suggested that certain adenoviruses, difficult to cultivate by standard cell culture techniques, might be an important cause of sporadic gastroenteritis in young children.

Organics. Fuchsbichler *et al.*[55] measured the uptake of 4-chloroaniline and 3,4-dichloroaniline in tomato, carrot, oat, barley, and wheat plants, and found uptake to be approximately proportional to the amount applied. Of the chloroaniline absorbed, 90 to 95% remained in the roots, except for carrots where about half was translocated to the upper parts of the plants. Johnson [56] argued that the widespread use of land treatment of wastewater was likely to contaminate ground water with toxic organics, and suggested that greater

control must be exercised, unless the public was willing to treat ground water as it did water from surface streams.

Inorganics. Fowler [57] provided a brief overview of the 1978 International Conference on Environmental Cadmium, summarizing and recommending future research in the areas of analysis methods, release into the environment, occurrence and mobility in nature, kinetics and metabolism, effects and dose-response relationships in humans, and effects and dose-response relationships in animals. A cadmium flow model for the U. S. was developed,[58] involving sources, use patterns, waste treatment and recovery techniques, waste disposal options, and environmental flow mechanisms. The model was used to simulate 10 years of cadmium environmental flow, fate, and human exposure for a variety of use patterns and treatment and disposal options. In another paper [59] the human dietary and respiratory exposure to cadmium was enlarged upon.

The results of an experiment on feeding young swine 50% Chicago wastewater sludge (83 ppm cadmium in diet), standard diet with cadmium chloride (83 ppm cadmium in diet), and standard diet alone (control) were reported by Osuna *et al.*[60] Both experimental diets depressed feed consumption and growth, the cadmium chloride diet (but not the sludge diet) produced iron-deficiency anemia, the sludge diet caused minor liver histopathological changes and acute cholangitis, and both diets raised liver and kidney cadmium concentrations (less on sludge diet). Sharma *et al.*[61] fed cows, swine, and chickens cadmium-chloride-supplemented diets containing 0.2 to 0.3 (control), 1.9 to 2.4, and 10.1 to 13.1 ppm cadmium, and found dose- and time-related increases in liver and kidney cadmium, no increases in bone, milk, or egg cadmium, no increases in cow and swine muscle cadmium, and a slight increase in chicken muscle cadmium (after 6 months of exposure only). It was concluded that although food-producing animals may act as an effective filter of cadmium in the case of an environmental increase of this metal, consumption of visceral organs from such animals may pose a hazard.

Hyde *et al.*[62] conducted 4 years of field studies and a 1-year greenhouse study to evaluate the effects of trace metals on crops in sludge-amended soils. The availability of cadmium to plants decreased with time as a result of chemical reactions which reverted cadmium to less soluble forms. Cadmium in corn grain was no different from controls even when annual additions of sludge amounted to cumulative cadmium added of 6.1 kg/ha in the calcareous soils. Agricultural management techniques were presented which effectively controlled the entry of heavy metals into the crops. Bingham [63] summarized the results of his greenhouse and laboratory experiments on factors influencing uptake and accumulation of cadmium by food crops, finding tolerance to cadmium and its distribution within the plant to be highly crop-specific, fruit and seed tissue to contain less cadmium than leaves, and uptake to be dependent on the cadmium concentration of the soil, especially free Cd^{2+} concentration. Blessin and Garcia [64] studied trace elements getting into the human food chain by transmission to corn grown on sludge-treated stripmine land, finding lead to be lower in corn grain from sludge-treated land, and cadmium to be double that for untreated corn, although all individual values were within the ranges found in commercial market samples. Trace element levels in commercial corn food products were also reported. Dorn [65] reviewed the subject of cadmium in the food chain, with the purpose of presenting the status of present knowledge on cadmium sources, levels in animal feeds, and intake, absorption, and distribution in the body of food-producing animals. Uthe and Chou [66] fed rats diets with equivalent levels of cadmium in the forms of lobster digestive glands, pork liver and kidney, and inorganic cadmium chloride, and found uptake from the organic forms to be only about half that from the inorganic form. Miller and Boswell [67] fed rats 50% turnip greens grown on industrial-type-sludge-treated soil, and found elevated liver and kidney cadmium. This result was in contrast with normal levels of cadmium found previously in rats fed 80% grain of corn, sorghum, and soybean grown on sludge-treated land. Fox *et al.*[68] studied the effects of nutritional factors on the metabolism of dietary cadmium in Japanese quail at levels similar to those of man, and showed that relatively small dietary changes could markedly affect tissue levels of cadmium and that a low intake of zinc might increase the risk of dietary cadmium exposure. Kostial *et al.*[69] studied cadmium metabolism in rats, and concluded that the young might be at a special risk at the same level of environmental cadmium exposure because of high oral cadmium toxicity at this age, which was most likely because of a high cadmium retention in the gut. Similarly, in experiments with mice, Engström and Nordberg [70] found decreasing intestinal absorption

of cadmium with increasing age, as well as higher intestinal absorption with cadmium pretreatment, and longer whole-body retention of absorbed cadmium with cadmium pretreatment and subsequent treatment. The authors hypothesized that the effects, that is, increased absorption and retention, of pre- and post-treatment with cadmium was a result of the induction of synthesis of the binding-protein metallothionein.

Baker *et al.*[71] found significant accumulation of cadmium in kidney and liver, but not muscle, of voles fed diets containing sludge-fertilized corn or sorghum. In studies with broiler chicks and laying hens, a 3-ppm cadmium diet caused cadmium accumulation in kidney and liver and a nonsignificant doubling of muscle cadmium. In 12 months of feeding laying hens, there was no transfer of cadmium to eggs. It was concluded that animal dietary levels of cadmium of less than 1 ppm from the use of wastewater sludge on land might, over a period of time, cause a significant accumulation of cadmium in animal tissues. Hinesly *et al.*[72] studied the effects of feeding corn grain produced on sludge-amended soil to pheasants and swine, and found levels of cadmium to be increased significantly in the liver and kidney. However, the maximum levels observed in tissues were not any higher than those reported by others in similar tissues from the same animal species reared under native or normal conditions. They concluded that levels of cadmium in liver and kidney tissues varied by animal species and age of animal as well as with dietary levels of the element, and where animal organs were an important part of human diets, animal age and species might be more important considerations for limiting cadmium in foods than amounts ingested by the animals as a constituent of grain from healthy plants. Muscle tissues from the animals remained unchanged in cadmium content. Hansen and Hinesly[73] fed corn grown with high-cadmium (200 ppm) industrial sludge to swine, and found increased kidney cadmium, decreased liver iron, and minor changes in liver microsomal oxidases and red blood cells, but no interference with swine performance. Swine foraging on the corn plots ingested considerable amounts of sludge soil and accumulated significantly higher concentrations of kidney cadmium. At lower rates of sludge application, the swine outperformed those foraging both on control plots and those receiving heavy sludge applications in terms of weight gain, in-utero piglet survival, blood hemoglobin, and tissue iron concentrations. Glaser *et al.*[74] studied cadmium toxicity in sheep, concluding that a daily intake of 30-ppm cadmium in food could be tolerated by sheep without subclinical biochemical-toxicological damage. The effect of cadmium chloride on the yield of chromosome aberrations in the bone marrow cells of mice was studied by Vilkina *et al.*,[75] who found no mutagenic effect. In a study of cadmium effects on rats, Whanger[76] found cadmium exposure caused a marked depletion of iron in liver and kidney; the addition of selenium to the diet decreased cadmium accumulation in liver and kidney (but increased it in testes); dietary ascorbate (vitamin C) decreased cadmium accumulation; there was no difference in absorption or accumulation of low-level cadmium from food as opposed to water; and cadmium did not contribute to hypertension. In a study on humans, no relationships were observed between blood or hair cadmium levels and blood pressure. The effects of dietary cadmium on rhesus monkeys was studied by Nomiyama *et al.*,[77] who concluded that the critical cadmium concentration in the renal cortex was approximately 380 μg/g wet weight for low molecular weight proteinuria, and approximately 470 μg/g wet weight for proteinuria, glycosuria, and aminoaciduria. These values are in contrast to the critical concentration of 200 μg/g in current use for man.

EPA and ORNL produced a document[78] tabulating detailed information on cadmium in foods published since 1940 and summarizing estimates of dietary intake of cadmium in the U. S. and foreign countries. Most foods contained low concentrations of cadmium, generally less than 0.050 mg/kg, wet weight. Concentrations of cadmium were greatest in shellfish, leafy vegetables, potatoes, grain and cereal products, and selected animal organs, particularly kidneys and liver. The use of cadmium-containing phosphate fertilizers increased the concentration of cadmium in some, but not all, plant foods. Based on the tabulated data, the average daily U. S. dietary intake appeared to lie within the range 30 to 50 μg, 42 to 88% of the recommended total tolerable limit of 400 to 500 μg/week. Excessive cigarette smoking could contribute as much, or more, cadmium as dietary sources. It was concluded that every reasonable effort should be made to limit further increases of cadmium in the environment. With respect to agricultural soils, such a policy implies careful control of the use of fertilizers and sludges

that contain high concentrations of cadmium. Kjellström [79] reported on a 3-year cooperative research study carried out in Japan, the U. S., and Sweden regarding comparability of analytical methods for cadmium, daily intake of cadmium via food, daily amount of cadmium in feces, concentrations of cadmium in different tissues, and the body burden of cadmium, urinary excretion of cadmium, and cadmium concentrations in blood. It was found that the cadmium intake via food among adults was about 35 μg/day in Japan (Tokyo) and about 17 μg/day in the U. S. (Dallas) and Sweden (Stockholm), and varied with age in a way similar to calorie intake. Kowal *et al.*[80] summarized the normal levels of cadmium in diet, urine, blood, and tissues from population samples in Chicago and Dallas, and found that the average daily intake of cadmium in food was 13 to 16 μg/day, and higher for males than females. Spencer *et al.*[81] conducted metabolic balance studies of cadmium, copper, manganese, and zinc in adult males, and found a mean dietary cadmium content of 32.9 μg/day. High levels of urinary cadmium, appearing to conflict with those found in the literature, were also measured. Nordberg and Kjellström [82] briefly described their metabolic model describing the flow of cadmium among eight body compartments in man, and confirmed the assumptions of a very long biological halftime in other tissues and the small excretion of cadmium via bile. Tsuchiya *et al.*[83] investigated the relationship between cadmium exposure and urinary β_2-microglobulin, and found them associated, but found age to be correlated more closely with urinary β_2-microglobulin. There did not appear to be a no-effect level of cadmium dose in elevating urinary β_2-microglobulin. Nogawa *et al.*[84] investigated the relationship between urinary cadmium concentration and several indices of renal effects of cadmium exposure, and concluded that urinary cadmium concentration could be useful in evaluating the renal effects in exposed people. Ellis *et al.*[85, 86] described a neutron-activation method for measuring absolute amounts of cadmium in the left kidney and concentrations of cadmium in the liver of living human beings. They found that cadmium body burden of smokers to be almost double that of nonsmokers. Roels *et al.*[87] used this technique to examine 309 male workers in two Belgian cadmium-producing plants. The cadmium concentration in urine and the urinary protein excretion were measured by standard techniques. It was concluded that the critical level of cadmium in the renal cortex was 200 to 250 μg/g, that the critical level of urinary cadmium was 10 to 15 μg/g creatinine, and that in the U. S. and Europe 95% of the middle-aged population were at least a factor of 5 below this critical level. A series of brief papers [88-90] appeared concerning the public health risk posed by high levels of soil cadmium (60–998 ppm) from an old zinc mine in the village of Shipham, Somerset, England. The villagers were advised to stop smoking, and avoid eating locally grown garden produce, which had high cadmium levels. *In vivo* neutron activation analysis showed the people to have five times higher liver cadmium concentrations (11 ppm) than controls. Although they showed no signs of cadmium toxicity, their liver cadmium levels were thought to correspond with kidney cadmium levels, which would produce early signs of renal tubular dysfunction. High zinc exposure may have ameliorated the effects of cadmium by decreasing the latter's absorption and toxicity. Twenty-two of 31 residents had raised blood-cadium levels, and some had hypertension and biochemical evidence of renal tubular damage, that is, increased urinary β_2-microglobulin. However, the method of sampling was criticized, the significance of hypertension challenged, and the validity of the data analysis questioned. Sandifer *et al.*[91] examined 49 golf course workers chronically exposed to cadmium-containing fungicide, and found no evidence of impaired renal function or hypertension. Bremner [92] reviewed the toxicity of cadmium in animals and humans as influenced by interactions with essential elements (calcium, iron, zinc, copper, and selenium), the low molecular weight metal-binding protein metallothionein, protein, age and milk diet, and vitamins. He concluded that any attempt to define permissible levels of intake of cadmium must take into consideration the variation in the nutritional status of the population, and that future estimates of permissible levels of intake of cadmium, and of other toxic elements, should be based on the expected response of those who would be most affected by exposure to the metals, that is, people with a poor nutritional status, the young, and the pregnant.

An extensive assessment of the health and environmental impacts of lead and its compounds appeared,[93] concluding that food may account for 60 to 90% of lead intake for the general population. Much of the lead was inadvertently introduced during food process-

ing, primarily from the lead solder used in food cans; no other lead limitation considered appeared to offer as great a potential benefit to the entire population as a reduction of lead in canned foods. Drill *et al.*[100] reviewed the sources of human exposure to lead, at least half of whose uptake usually came from the lead in food. Needleman *et al.*[101] examined lead levels in the deciduous teeth of children, and found that lead exposure, at doses below those producing symptoms severe enough to be diagnosed clinically, appeared to be associated with neuropsychologic deficits that might interfere with classroom performance. Concern for human exposure to lead was expressed by Lin-Fu,[102] who concluded that a reexamination of the concept of lead poisoning was needed, and that future hazardous lead exposure must be prevented.

RENOVATED WATER

Swayne *et al.*[103, 104] examined the proportion of wastewater in surface waters used as drinking water supplies in the U. S., and found maximums of 20 and 5 million persons to be exposed to wastewater exceeding 10% and 100%, respectively, during low-flow conditions. This data can be used as an estimate of existing indirect wastewater reuse for potable purposes. An ongoing epidemiological and toxicological study of the health effects of recharging ground water with renovated wastewater at Montebello Forebay, Calif., was described by Nellor.[105] Andelman[106] analyzed reclaimed process wastewater for reuse from a poultry processing plant, and found, with only a few exceptions, that the mean and maximum concentrations of the various measured constituents met existing standards for potable water. In the rare cases when they did not, such as maximum value marginally exceeding a recommended limit for sulfate, even with direct consumption of the water there would be no danger to human health. Crook and Spath[107] reviewed wastewater reclamation in California and its potential health effects, concluding that direct potable reuse of wastewater should be deferred until enough scientific evidence was available to assess adequately the potential adverse health effects.

Parasites, bacteria, viruses. Brewer and Carmichael[108] characterized the microbial populations of granular activated carbon (GAC) filter systems, and concluded that bacterial populations adhered and accumulated on GAC filters during water filtration. This buildup may lead to the potential accumulation of toxic products, that is, endotoxins, which could flush through the filter when the filter capacity was reached. Wolf *et al.*[109] studied the endotoxin content and pyrogenic response in rabbits of granular activated carbon filtered waters; GAC-filtered secondary effluent contained 6 to 250 μg/l and yielded pyrogenic responses in 18 of 20 trials, while 27 different GAC-filtered water supplies had levels of 1.2 to 25 μg/l and no pyrogenic response. No relationship was observed between endotoxin content and pyrogenic response. Good correlations were observed between endotoxin content and standard plate count and total coliform count in nondisinfected AWT effluent, but poor correlations observed between endotoxin content and bacterial counts in disinfected AWT effluent.[110] The subject of the health significance of viruses in drinking water was briefly reviewed by Smith,[111] who encouraged further research in the field. A report of the American Water Works Association Committee on Viruses in Drinking Water[112] reviewed the state of the art of environmental virology as applied to water treatment criteria. Among the conclusions were that treatment criteria for the direct production of potable water from wastewater must be carefully developed, and that the viral quality of ground water must be continually assessed as a result of increased land application of wastewater and sludge. Dryden *et al.*[113] compared virus removal in several advanced treatment systems, consisting of coagulation-sedimentation-filtration, direct filtration, and carbon adsorption followed by disinfection with either chlorine or ozone in each system. Both natural viruses and attenuated Polio 1 seed virus were used. At an average combined chlorine residual of 10 mg/l, all systems produced approximately 5.2 logs of seed virus removal.

Organics. Preliminary results were reported by Pahren and Melton[114] on a study of the mutagenic activity and trace organics content of solvent-extracted reverse-osmosis concentrates of advanced wastewater treatment plant effluents. No relationship between mutagenic activity and either identified organics or functional groups in a sample was evident. However, mutagenic activity showed some very general relationships with treatment and concentration methods, that is, high with chlorination as final unit operation, low with either activated carbon or reverse osmosis as the final step, higher with cellulose acetate than nylon no concentration membrane, and higher with acidic methylene chloride than pentane or

neutral methylene chloride. Gruener and Lockwood [115] used selected *in vitro* assays to evaluate the mutagenic and carcinogenic potential of a concentrated, recycled water sample. They found that the sample induced mutagenicity in hamster lung cells and cellular transformation in human embryonic lung fibroblasts, even though negative in the Ames test. *In vivo* toxicological tests done in mice, after exposure up to five months to TOC levels up to about 1 000 times the expected human exposure, did not show any pathological changes.[116] Gruener [117] performed Ames tests on recharged ground water in Israel before and after ozonation, and concluded that mutagens were formed by the ozonation process. Bercz [118] discussed several screening tests which might be used for checking toxicity of waters, with emphasis on the Ames test. It was his view that the Ames test was valuable for orientation only, and should not be used alone for regulatory purposes. It could be used to predict which chemicals were not carcinogenic but had a probability of only 0.27 of finding a carcinogen among all Ames positives. Cody *et al.*[119] presented some examples of the use of a mammalian cell tissue culture assay for the assessment of quality of recycled wastewater. The general approach was adaptable to screening for toxicity of drinking water, wastewater, recycled water, and natural surface and ground water. Saxena and Schwartz [120] assayed, by Ames test, mutagens in wastewaters renovated by advanced wastewater treatment, and concluded that the physico-chemical and biological AWT methods studied were unable adequately to remove potentially hazardous chemicals from wastewaters. Saxena *et al.*[121] arrived at the same conclusion on examining the influent and effluent of a high-lime process AWT plant for mutagens. Organic wastewater concentrates from six treatment plants were tested by Rappaport *et al.*[122] for mutagenicity using the Ames mutagenic bioassay. Concentrates were prepared by passing 4-8 l of wastewater through two porous copolymer resins (Amberlite XAD-2 and XAD-7) in series. Mutagenic responses were observed in extracts from most of the plants tested. More activity was found in secondary and post-secondary extracts than in primary extracts. Fractionation of positive extracts showed the activity to be primarily in the basic and neutral fractions, while acid fractions were relatively inactive. All of the definitely positive samples were obtained from plants that treated mixed domestic and industrial wastes, while samples obtained from plants treating strictly domestic wastes were always negative or marginal. Based upon the adenylate metabolic pathway, a rapid method of detecting cellular toxicity using human platelets, neutrophils, and monocytes was described by Solomons,[123] and tested on wastewater concentrates. The technique was suggested for the detection of toxic compounds within hours of sampling, and, thus, as being applicable to modern water reuse technology. The feasibility of short-term shower and laundry water reuse by field Army units was evaluated, with reference to health effects, by Cogley *et al.*[124] None of the available data indicated that a toxic response was to be expected for soldiers using treated, recycled shower and laundry waters, and a human clinical trial of shower and laundry water reuse was recommended.

Inorganics. Seventy trace elements in four advanced wastewater treatment plant effluents were measured by proton-induced X-ray emission, and reported by Pahren and Ulmer,[125] to determine if any element that was not ordinarily checked might be present to a level which would be of health concern if the water were to be reused for potable purposes. It was found that trace elements which were not normally checked were either below detectable limits or not of a level to be of health concern.

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